**Do Laboratory-Based Methods to Identify Causes of Toxicity in Sediments Really Reflect Field Conditions?**

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Toxic sediments are of concern to regional, state, and tribal managers. While sediment toxicity tests are good indicators of toxicity in sediments, they do not provide information on the cause of toxicity. Chemical scans of sediments are expensive and do not give information on the bioavailability of compounds. Sediment Toxicity Identification and Evaluation (TIE) methods use both toxicity tests and chemical manipulations in an approach designed to identify specific cause(s) of toxicity (i.e., metals, organics, ammonia) in sediments. Identifying the cause of toxicity allows managers to design effective remediation methods, identify sources, and develop effective source tracking and reduction strategies. While the efficacy of TIE methods in the laboratory is fairly well demonstrated, the question of whether the same toxicant identified in the laboratory is causing effects in the field remains unanswered. In this study, a number of collaborators used different methods in a weight-of-evidence approach to determine whether laboratory TIE methods accurately predict field effects. A TIE performed on sediments collected from the Elizabeth River (VA) identified polyaromatic hydrocarbons (PAHs) as the major toxicant. Several lines of evidence indicated PAHs were the major toxic agents in the field as well. Concentrations of PAHs in Elizabeth River sediment were elevated relative to a nearby reference site. Chemical analyses of exposed bivalves indicate PAHs occurred in high concentrations in the bivalve tissue; concentrations of poly chlorinated biphenyls (PCBs), another potential toxicant, were below detection levels in the same tissue. The Comet assay, which measures DNA damage and is sensitive to PAHs, indicated adverse effects in caged bivalves in the Elizabeth River relative to those from a reference site. In addition, *Fundulus heteroclitus* exposed to extracts of Elizabeth River sediment responded similarly to fish exposed to PAH model compounds and differently from fish exposed to PCB model compounds. Our final line of evidence was the response of benthic organisms exposed to Elizabeth River sediments and then exposed to ultraviolet (UV) radiation. UV radiation causes a toxic diagnostic response unique to PAHs. The aggregation of these various lines of evidence supports the conclusion that PAHs are active in both laboratory and field-exposed organisms and that laboratory-based TIE methods reflect field conditions. This research supports the Agency's need

to identify stressors in waterbodies/watersheds by field validating methods that link sources, stressors, and effects.

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